SAFEGUARDS OF THE HEART-BEAT.

BY HENRY SEWALL, Ph.D., M.D.,
PROFESSOR OF PHYSIOLOGICAL MEDICINE IN THE DENVER AND GROSS MEDICAL COLLEGE, DENVER, COLORADO.

THE student of disease loses nothing in accuracy and may gain much in comprehension by pausing now and then to contemplate the principles involved in phenomena that are trite and familiar. Thus, to preserve the equilibrium of the upright body there is a single physical condition which is all-essential, namely, that a vertical line dropped from the centre of gravity of the body shall pierce the area circumscribing the feet. Let the frame be bent to any extent in one direction, equilibrium may still be preserved by a definite mechanical displacement in the opposite. Whether the area of support be large or small, the distortion of the figure great or little, the body will still stand provided its centre of gravity remain within the area of support.

What is this but *compensation*, which secures the realization of a purpose by maintaining the balance of opposing forces? Turning to the almost infinitely complex system of units of matter and energy whose equilibrium we know as Life, we always find that a departure from a mean in one direction is accompanied by a corresponding deviation in another; so that the terms "health" and "disease" find their best definition, not according to some specific adjustment of structure and function, but according to the efficacy of the balancing of ever-shifting forces by which life is maintained. We are wont to think of compensation in vital activity as some unusual adjustment to meet a critical functional failure; whereas, probably every physiological feat accomplished manifests the balancing of equal and opposite forces. The principle here involved is perhaps the main factor in biogenesis, but, nevertheless, students are prone, especially on the clinical side, to neglect its application.

While we must ascribe most adjustments of living structures to environment to slowly acting natural selection, the history of a single individual may prove the intimate dependence of growth upon physical conditions. Thus, Babak[1] has shown that the external gills of amphibian larvæ grow luxuriantly when the animals are kept in water poor in oxygen, but attain progressively smaller dimensions when the oxygen pressure is increased; that is, the oxygen-absorbing organs wax or wane in volume in order that the tissues they supply may receive the needed complement, no more, no less, of the vitalizing element. To the same physiological category belongs the familiar fact that the pupil of the eye dilates in the dark and contracts in the light, whereby the sensory cells of vision escape overstimulation and appreciate feeble illumination.

---

[1] Ref. in Biophysikal. Centralbl., December, 1907, 191.

Sherrington has demonstrated that even a simple motor impulse to a skeletal muscle involves a simultaneous inhibition of the tonus of the antagonistic muscle group. The mechanisms of the body are probably all radically imperfect when measured by the demands we make of artificial instruments. Yet it will doubtless be found in every case that each dereliction from mechanical perfection is so balanced by compensations that the range of usefulness of the organ to the body is thereby positively enhanced. For example, the eyeball is commonly considered to have its analogue in the photographer's camera. But its imperfections as such an instrument would make it useless in the arts for the simple reason that its whole surface is permeable to light. But light that enters the eye by any part other than the pupil must have penetrated many bloodvessels and become red in color. Under ordinary conditions of vision, then, the retina is bathed in diffuse reddish light, except for a spot in the centre upon which falls the white light which has entered through the pupil. According to the laws of physiological contrast, the sensitiveness of the retina in this area must be kept ever keen for the complementary color to that which surrounds the central spot, and that color is green. As I have already pointed out,[2] the psychic effects of the verdure of Nature, the uncloying joy of its greenness, find herein a physiological basis. Moreover, it has also been demonstrated that acuity of vision, which measures the optical fitness of the eye, is preëminently keen when objects are viewed in a light of greenish tinge. When, in a sensory apparatus, we find that the most hopeless physical defects are turned into the finest uses of the instrument, we may grant, without prolonging this line of analogy, that there is overwhelming *a priori* probability that every vital organ must be abundantly provided with efficient means of circumventing disaster from the overstrains which the exigencies of living necessarily more or less habitually entail.

The physiologist has no compunction of doubt in describing the heart as essentially a force pump, the continuity of whose current implies the efficient closure of the auriculoventricular valves. It is easy to demonstrate in the excised, normal heart that the perfection of valve action should leave nothing to be desired. Indeed, on forcing water into a human left ventricle, having somewhat fatty walls, through a tube tied in the aortic orifice, I have caused the heart wall to burst while the mitral valve refused to leak. When one considers the limited range of dilatability of the cardiac cavities, the extraordinary variability of the inflowing blood current, the readiness with which the heart may be overstrained by increase of the diastolic blood pressure, and the irretrievable disaster entailed by prolonged asystole, it would seem on *a priori* grounds that there must be some adaptation of the cardiac mechanism, not obvious in its functional structure, by which the perils of these conditions may be averted. This is practically the conception at the basis of the epochal work of T. Wilkinson King,[3] who, in 1837, published "An Essay on the Safety-valve Function on the Right Ventricle of the Human Heart; and on the Gradations of this Function in the Circulation of Warm-blooded Animals." King demonstrated on a large series of mammals that when the right ventricle is distended with water introduced through the pulmonary artery, there is a reflux of fluid into the right auricle. This insufficiency of the tricuspid valve is brought about by stretching, under pressure, of the yielding wall of the right ventricle to such an extent that the valve flaps are prevented from coming into approximation. Clinicians are daily in contact with examples of "relative valvular insufficiency" occurring in patients having dilated hearts. But when, according to clinical symptoms usually relied upon, there is demonstrable leakage of the tricuspid valve, it seems a reproach of Nature to describe this as a safety mechanism, for the patient is already in desperate straits. From clinical observation as well as theoretical considerations I have long held the view that overdistention of the ventricles is guarded against by compensatory insufficiency of the auriculoventricular valves which is so frequent as to be properly classed among physiological phenomena.

---

[2] On the Physiological Effects of Light which Enters the Eye through the Sclerotic Coat. Jour. Phys., 1884, 132.

The physical conditions of the cardiac circulation are such as presumably to permit extensive regurgitation from either side of the heart without notable functional disturbance. Each auricle receives its blood stream under a low head of pressure from a reservoir having a capacity many times as great as the chamber which it is to supply. The walls of these reservoirs, the pulmonary veins and capillaries for the left auricle, and the venæ cavæ and their branches for the right auricle, are peculiarly extensible. These channels are capable of accommodating, probably without very notable increase of intravenous pressure, an amount of blood much in excess of that usually contained therein. The term "reservoir" which has of late frequently been used to designate these vascular territories is really descriptive of their function. It is to be expected that any backflow from the heart which does not so raise the venous pressure as to impede the circulation in the vital organs, may be tolerated without producing physiological disturbance. It may, perhaps, also be assumed that at the beginning of a given systole, the regurgitation of an amount of blood which is very small in proportion to the capacity of the venous reservoirs, might save the heart from dangerous overstrain; and toward the end of systole such backflow could prevent accumulation of residual blood. It is obvious that these considerations present a theoretical means of protecting the heart against overstrain during a temporary excess of inflow without in any way interfering with the work of other organs; in other words, such regurgitation from the ventricles would represent a true "safety-valve" action of the auriculoventricular leaflets. As will be pointed out later, it is to be expected that such valvular insufficiency would altogether escape ordinary observation because the amount of blood thus injected into the auricles and veins is not sufficient sensibly to distend them. But when the backflow through the auriculoventricular valves persists, the excess of blood entering the heart over that leaving it is stored up in the auricles and veins, and leads to a progressive increase of venous pressure. In such an extremity the veins are engorged and may pulsate visibly like arteries with contraction of the ventricle, because the elasticity of their walls has been pushed to the limit. The heart is now permanently dilated; the auricles may be fixed in paralytic distention, and the atrial rings are so stretched that their valves are relatively insufficient. Nevertheless, the disastrous march toward venous plethora and arterial ischemia may be checked at any point whenever the forces of compensation are able to equalize the rate of inflow and outflow of the heart. Wonderful as it seems, the physiological demands of the body may be satisfied indefinitely by a circulation maintained by a pump with radically defective valves.

---

[3] Guy's Hospital Reports, 1837, ii.

Clinical observers, for the most part, appear to regard the occurrence of tricuspid insufficiency as a late link in the chain leading to heart failure—an event manifested by engorgement and pulsation of the central veins, enlargement and pulsation of the liver, and a well-marked systolic murmur audible over the precordium with maximum intensity in the tricuspid area. Gibson, however, is quoted by Babcock[4] as thus characterizing tricuspid regurgitation: "It is incomparably the most common of valvular lesions, and the reason this fact is not brought out in statistics upon the relative frequency of valve defects is to be found in the circumstance that incompetence of the tricuspid valve does not in itself seriously impair the general course of the circulation, and it is therefore often found among those, who, although under treatment for various affections, have no cardiac symptoms. It accordingly escapes observation unless especially sought for." Babcock deprecates this statement of Gibson's which is, however, quoted here because it expresses exactly the conclusions to which my own clinical studies have led. The class of cases which affords the evidence for this belief is one in which there are usually no subjective symptoms and few of the classic signs of cardiac disorder. In fact the subjects have not heart disease, as a rule, but only the functional expression of cardiac overstrain. There is often even no complaint of shortness of breath on exertion. A systolic murmur, usually faint and soft, with much the quality of the normal inspiratory sound in the lungs, is often heard with greatest intensity along the lower left border of the sternum. But the auscultatory signs in this region are too various and uncertain in origin to permit of diagnosis by the murmur alone. Careful outlining of the heart by percussion usually shows the area of dulness to be somewhat increased to the right, but this sign is by no means distinctive of valvular insufficiency.

---

[4] Diseases of the Heart and Arterial System, 1903, 343.

In an admirable series of experiments, Roy and Adami[5] long ago studied the time relations of movements in various parts of the dog's heart. They compared the movements of the cardiac wall with those of the papillary muscles by means of tracings obtained from levers attached to hooks, one of which was plunged into the surface of the ventricle and the other inserted through the edge of a mitral valve.

Their records show that at the beginning of ventricular systole, during the period of "rising tension," the edge of the valve is forced somewhat into the cavity of the auricle, but that the terminal act of ventricular systole is marked by a sharp downward jerk of the valve as the wave of contraction invades the papillary muscles; and it is, according to them, this sudden increase of tension which throws open the aortic valves. Forewarned of the danger of depending upon *a priori* arguments, we may still assume provisionally that, were the wave of contraction to fail to involve the papillary muscles, these bands would be stretched like elastic cords, and, as the ventricular parietes approximate in systole, a leakage through the up-pushed valve should be expected. It is evident that the period in the cardiac cycle at which such regurgitation would occur might either cover the whole period of ventricular contraction, or be limited to the early phase thereof in case the contraction wave progressed with unusual slowness, or it might be restricted to the end of the ventricular outflow in case the contractile tonus first abandons the papillary muscles. Tracings of the venous pulse suggestive of the latter condition are obtained with great frequency. My conception of functional contraction failure of the papillary muscles was suggested by the analogy of results obtained many years ago in experiments upon the skeletal muscles. When a lever is laid over each end of a frog's muscle, which is fixed at one extremity and rests upon a horizontal support, the contraction wave, started at one end of the muscle by a submaximal stimulus, is found to diminish progressively in height as it traverses the fibers. As the stimulus is weakened the wave involves less and less of the length of the fibers. When a third lever, in connection with the free end of the muscle, registers its shortening, it is found that the length of the latent period of contraction varies inversely with the strength of the stimulus; that is, the greater the length of the inert elastic tissue that is interposed between the contracting moiety of the muscle and the lever, the more time it requires to bring the inactive part of the muscle into tension sufficient to lift the weighted lever.

---

[5] Heart-beat and Pulse Wave. Practitioner, 1890.

The analogy between these results and the facts of cardiac activity is not far to seek. Physiological experiment on the excised heart muscle shows not only variability in the rate of progress of the contraction wave, but, at least in cold-blooded animals, an extraordinary tendency for the contraction, started at one end of a bundle of fibers, to die out before it reaches the other. The clinical phenomenon of "heart block" is sufficient evidence that somewhere in the contractile chain a conducting link is defective.

Students of the heart-beat have generally been impressed with a something like specificity in the functions of the papillary muscles. In another place[6] certain evidences in this direction are thus presented: "J. G. Adami,[7] summarizing the results of experiments made by Roy and himself on the dog's heart, speaks of the contraction of the papillary muscles as 'virtually independent' of that of the ventricular wall. 'The more we have studied the tracings obtained under various conditions, the more we have been led to conclude that the moment when they begin to contract is not primarily dependent upon the moment of commencing ventricular contraction. We find, for example, that an overdose of liquor strychninæ may lead to complete asynchronism between these two components of the ventricular action; or, again, there may be a ventricular systole unaccompanied by papillary contraction, or vice versa. Fenwick and Overend[8] studied the contractile movements of the papillary muscles and apex of the excised heart of the rabbit. They found that the papillaries began their contraction later than the apex, and they state: 'We never observed a difference as regards the commencement of the curves of less than one-twentieth of a second. The papillaries, however, speedily lag more and more behind the ventricles until they cease altogether.'''"

Haycraft and Paterson,[9] in repeating these experiments, find that in the fresh heart the papillary muscles contract simultaneously with the ventricle. Later the papillary muscles may begin to contract before or after the ventricular wall. Fenwick and Overend continue: "It is well known that these muscles [the papillaries] are particularly prone to all forms of structural degeneration, and that their condition constitutes the most delicate index of the general state of cardiac nutrition. In fact they might be termed the 'cardiac extremities,' since, from their single attachments to the ventricular walls, they must necessarily derive their entire blood supply from the nutritive vessels which enter at their bases. In short, their circulation must be of a terminal character. It is, therefore, obvious that the *musculi papillares* are at the outset even structurally predisposed to exhaustion and degeneration, and any condition which tends to lower the nutrition of the heart as a whole must affect them with a magnified degree of intensity."

---

[6] On the Clinical Relations of the Papillary Muscles of the Heart. Philadelphia Monthly Med. Jour., September, 1899.

[7] On the Action of the Papillary Muscles of the Heart. Proc. Cambridge Philosoph. Soc., vii, part II.

[8] Report on the Contraction of the Papillary Muscles, and its Relation to the Production of Certain Abnormal Cardiac Sounds. Brit. Med. Jour., 1891, i, 1117.

[9] The Time of Contraction of the Papillary Muscles. Jour. Phys., 1896, xix, 262.

In reflecting on the feasibility of obtaining experimental evidence of functional tricuspid insufficiency, it seemed probable that clinical studies of the jugular pulse might furnish the desired facts. From the very nature of the conditions assumed to underlie such circulatory failure, it is manifest that the clinician and not the physiologist can find ready at hand appropriate material for observation. At the very outset of these studies I was astonished at the unexpected wealth of material presented in the persons of patients whose hearts would pass as perfect to ordinary clinical examination. One easily becomes convinced that the vascular movements of the neck are concerned much more prominently with changes in the veins than in the arteries. The observations can usually only be made while the subject is in the recumbent position. When this posture is assumed, a rapid, more or less, dicrotic undulation of the skin above the inner ends of the clavicles is common and signifies variations in the volume or tension of the great vessels beneath. But, to one who has not definitely investigated the subject, it will cause surprise with what frequency the external jugular veins themselves become prominent and actively pulsate.

With a stethoscope applied to the heart, it is plain in this class of "normal" subjects that the swelling of the external jugular vein usually begins just before, and reaches its acme during the first sound of the heart; it is common, perhaps usual, for the blood wave to distend the vein with a dicrotic motion. When a jugular vein is clamped by finger pressure high in the neck, it drains more or less completely. A pulsatile filling of the vein under these conditions seems to postulate a true systolic regurgitation from the auricle or ventricle rather than a mere rhythmic interruption of the stream which normally traverses the vein. But more commonly it occurs that the swelling and pulsation of an external jugular vein completely disappear when the vessel is occluded above; therefore, the important conclusion is deduced that *venous pulsation does not necessarily predicate the translation of blood mass from below, but only that of a mechanical impact.* Not infrequently, with each heart-beat the neck swells laterally along the course of the carotids. That this slow wave is due to engorgement of the internal jugular trunks and not to arterial pulsation seems proved by the fact that it ceases if the fingers are pressed gently down behind the inner end of the clavicle; at the same time the external jugular vein on the same side becomes turgid and its pulsation ceases. The pressure has simply blocked the communication between the jugular branches and the heart without interfering with the carotid blood flow, which still may transmit its abrupt pulse wave to the tissues.

Graphic tracings may be made of these various pulsations. When the transmitting tambour is placed over the carotid artery under the angle of the jaw, the tracing obtained closely resembles the sphygmogram of the radial pulse. When the tambour is applied to the area of undulation above the clavicle, the record may still have the characters of the arterial sphygmogram or it may take on quite a new form, that of the venous pulse.

According to Mackenzie[10] the occurrence of a venous pulse always indicates a certain degree of cardiac insufficiency and "limitation of the field of physiological response," recovery from which depends upon the degree of overstrain to which the myocardium has been subjected. In certain cases it can be noticed that a typical venous pulse tends to assume the arterial form after the subject has taken a few deep breaths. With the registering apparatus usually employed, the venous pulse is found to be made up of a group of three characteristic waves (*v*, *a*, *c*, Fig. 1), a special discussion of which is not intended here.

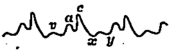

FIG. 1.—Typical venous pulse. The depression *x* marks the extreme relaxation of the auricle, and *y* the corresponding state of the ventricle.

The *a* wave is admitted to be synchronous with the contraction of the auricle and to be caused by it. The *c* wave is synchronous with contraction of the ventricle, though the cause of its formation is in dispute. When the levers registering the venous and the carotid pulse are placed in the same vertical line on a (travelling) smoked paper, it is found that the *c* wave begins about the period of outflow into the aorta, as marked by the ascent of the carotid tracing. The *v* wave, in the class of cases here under discussion, usually begins about the end of the period of outflow from the heart, marked on the carotid tracing by the dicrotic notch. The origin of the *v* wave is ascribed by Mackenzie to the rising pressure within the auricle due to the constant inflow of blood during the ventricular systole. The sudden decline of the *v* wave is dependent upon the emptying of the auricle into the distending ventricle, after completion of ventricular systole. Among patients presenting themselves for examination on account of a wide range of functional disorders, I have been struck with the uniformity with which evidences of cardiac insufficiency could be distinguished, based both upon the nature of the symptoms and the character of the venous pulse. I have been led to believe that functional insufficiency of the tricuspid valve is of frequent occurrence in such cases, and that it occurs without extraordinary dilatation of the heart. Objective evidence of such regurgitation is furnished by certain forms of the venous pulse. Thus, in Fig. 2, it is seen that the *v* wave has a double crest, or, rather, the wave *v* which begins just at the moment of closure of the aortic valves, as determined by the dicrotic notch in the lower tracing, is immediately preceded by a wave which is completed during the last moments of ventricular outflow. At this time the auriculoventricular valves should be firmly closed and there is no apparent reason why the gradual increase of auricular diastolic pressure, as marked by the *v* wave, should be interrupted.

---

[10] The Study of the Pulse, 1902.

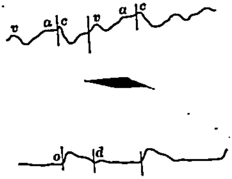

FIG. 2.—From an anemic girl aged fourteen years. The upper tracing is that of the right internal jugular pulse, showing a double crest to the *v* wave. The lower tracing is from the carotid artery; *o* is the beginning of ventricular outflow, and *d*, at the dicrotic notch, marks the cessation of outflow. Events on the same vertical line in the two tracings are simultaneous.

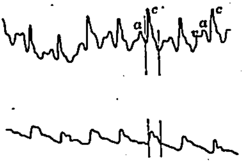

FIG. 3.—Tracings of the right internal jugular and left carotid pulses from an athletic boy aged eighteen years, who had overstrained his heart with exercise. Note the undulations on the descending limb of the *c* wave, which can hardly be due to instrumental defects.

But if we may assume that toward the end of ventricular systole the papillary muscles relax, it is obvious that the tricuspid valves might be lifted by the intraventricular pressure and allow the regurgitation of a certain amount of blood which might cause the first elevation of the wave *v*. It is obvious that the earlier in the systole of the ventricle the assumed papillary relaxation occurs, the earlier is the projected wave depicted upon the tracing; thus, in Fig. 3, the notch on the descending limb of the *c* wave apparently illustrates such a condition. Marked insufficiency of the papillary muscles occurring early in ventricular systole might readily so reduce resistance to regurgitation that the arterial outflow would cease and the semilunar valves be closed prematurely.

The well-known variations in the height and extent of the *v* wave which depict the progress of early cardiac insufficiency are readily explained by conditions like these. These graphic symbols have their symptomatic counterpart, and, in a rather limited experience, I have been gratified with the therapeutic results of treatment directed to the conservation of cardiac energy in such cases. Mackenzie exhibits many tracings which represent the double apex of the *v* wave. In his explanation of the double crested *v* wave, he attributes the portion preceding the notch, which coincides with the terminal period of arterial outflow, to the same accumulation of blood within the auricle, which, according to him, causes the usual *v* wave, while he assumes that the portion of the wave following the notch to be due to tricuspid regurgitation resulting from the resistance to backflow into the auricle suddenly falling below that offered to outflow through the pulmonary artery. Such an explanation would involve a shortening of the period of outflow, for which there seems no evidence, and, according to pulse tracings, an extreme prolongation of the period of ventricular contraction. According to the theory proposed in this paper, regurgitation might readily occur either before or following semilunar closure or during both periods. In cases of "premature systole," with the stethoscope applied over the heart, one frequently hears a short first sound, sometimes accompanied by an obscure murmur, followed quickly at times by a second sound of semilunar closure. The radial pulse wave belonging to the short ventricular systole is usually absent, but at the moment of systole there is a sudden bulging of the jugular bulbs, which forms a striking feature of the dyscrasia. An obvious explanation of the phenomenon is that the contractile wave which seizes the ventricular muscle fails to reach the distal extremities of the fibers; the resistance to outflow into the auricle is, therefore, less than that into the aorta and, accordingly, most of the ventricular content is regurgitated. The fact that the aortic resistance, and doubtless that in the pulmonary artery as well, rises steadily from minimal to maximal pressure between the beginning and the end of ventricular outflow, may possibly determine the failure of the weaker part of the cardiac muscle toward the end of systole. Hewlett[11] would probably class such regurgitations as examples of the "positive venous pulse" dependent upon simultaneous contraction of auricles and ventricles. Even if these two chambers do contract simultaneously, of which there has been no evidence in the examples studied by me, such synchronism of action should be attended by regurgitation from the ventricle, because its systole begins while the auriculoventricular valves are still open. But, as demonstrated by Mackenzie in his chapters on irregular heart action, the venous pulse in such cases can only be interpreted in the light of an exact conception of the relative time phases of auricle and ventricle, a subject which cannot be entered into here.

---

[11] The Interpretation of the Positive Venous Pulse. Jour. Med. Research, October, 1907.

Before concluding this important subject it is well to picture again the condition of hydrostatic pressure existing within the ventricle toward the end of ventricular systole. The whole mass of the organ is in strong contraction. The auriculoventricular valves are held down at an indeterminate level by the pull of the papillary muscles. Suddenly, now, the outflow through the pulmonary artery ceases and, *provided there remains in the ventricle a sufficient residue of blood*, there is just as suddenly imparted to the under surface of the auriculoventricular valve an excess of pressure proportional to the relief of strain which has been cut off by closure of the semilunar valves. It seems wholly probable that the valve should thereby be pushed more or less into the cavity of the auricle and impart to its contained blood an impulse. As has already been shown, such an impulse could give rise to a wave in the venous tracing without necessarily involving any considerable displacement of blood mass. It is most significant that the more nearly normal the subject in whom the venous pulse is recorded, the more exactly does the beginning of the *v* wave correspond with the moment of closure of the semilunar valves. Now let us conceive the course of events under conditions of weakness of the papillary muscles. The resistance to ventricular outflow increases as the systole progresses and arterial pressure rises. This heightened hydrostatic pressure is imparted to the auriculoventricular valve, producing a strain whose total value is dependent upon the surface exposed. It should be expected that, were the tone of the papillary muscles to lessen toward the end of systole, still more, were the valve to become insufficient, we would find the *v* wave of the venous pulse to take its origin at a variable period preceding semilunar closure; and any sudden increase of the impulse imparted to the auricular contents, such as might be caused by a jet of blood escaping from the ventricle, could very well cause a doubling of the *v* wave such as is actually found in tracings of certain weak hearts.

From an *a priori* point of view it would seem probable that a safety-valve action of the auriculoventricular mechanism would be especially liable to occur at the beginning of ventricular systole when the ventricle is most distended and its contractile substance at the greatest mechanical disadvantage. It would seem that any undue retardation of the wave of contraction might permit such stretching of the papillary muscles as to allow regurgitation of blood during the "period of rising tension." The *c* wave, which covers most of the period of outflow from the ventricle, should be modified by such regurgitation, but the factors entering into its production are still too uncertain to permit conclusions to be drawn from its change of form. It may be said, however, that in many of my tracings from cases of functional heart weakness, the magnification in the height of the *c* wave is very suggestive of valvular insufficiency early in systole.

The so-called safety-valve action of the auriculoventricular trapdoor, whatever its significance in the cardiac economy, is generally regarded as an attribute of the right ventricle alone. If there be any truth in the proposition advanced above, that insufficiency of the auriculoventricular valve may occur through stretching of inert papillary muscles at the time of ventricular systole, there seems no reason why the same events should not, under appropriate circumstances, attend the action of the left ventricle. The anatomical fact that the tricuspid valve is suspended to a thick, circular ring of muscle, while the larger anterior flap of the mitral valve is made by a prolongation of the aortic wall, would lead to the expectation that functional dilatation of the auriculoventricular opening of the right side might easily occur, but not of the left. Tracings of the jugular pulse seem to offer experimental evidence of events occurring on the right side of the heart. One might expect that the record of movements of the left auricle, which can be obtained by connections with a rubber balloon resting in the œsophagus, should furnish certain evidence of mitral regurgitation. Yet Rautenberg,[12] in his observations on this subject, expressly states that in well-compensated organic insufficiency of the mitral valve the tracings of pressure changes and movements of the left auricle are almost identical with those derived from the normal subject. Yet speculation, if sharply defined as such, seems entirely excusable on so important a subject.

The distresss of an enfeebled heart is, in general, determined by the strain imposed by an excess of blood in one or more of its chambers. It is manifestly important for the pathologist to know the normal variations of the intracardiac blood pressure and the reactions of the cardiac chambers thereto. There can be little doubt that the movements of respiration normally produce a rhythmic variation of the ratio of intracardiac pressure on the two sides of the heart, yet definite physiological observations on this important subject seem to be wanting.

It is admitted that the inflow of blood to the right auricle is accelerated with the movement of inspiration and retarded with that of expiration. In inspiration the vascular bed of the lungs is increased and the resistance opposed to opening the pulmonic semilunar valves is probably diminished; but the aspirating force of inspiration would presumably be still more effective in retarding the blood flow into the left auricle; therefore, during inspiration we should expect an overfilled right heart and an underfilled left heart. The mechanical effect of an expiratory movement is just the reverse, and during expiration the diastolic content of the right heart would be decreased, while that on the left would be increased. An alternation of heart strain on the two sides would presumably run parallel with the degree of filling. A comparison of the curves of intrathoracic and arterial blood pressure gives general support to the foregoing position;[13] but the temptation to build upon even the most plausible theoretical notions is counteracted by the advance knowledge that the only trustworthy conclusion is to be reached by exact experimentation.

---

[12] Die Registrierung der Vorhofspulsationen von der Speiserohre aus. Deut. Arch. f. klin. Med., September, 1907.

It is very desirable that we should have at hand means for the clinical estimation of the changing relations of heart strain. There is reason for supposing that such a means is to some extent provided in the *physiological* reduplication of the heart sounds.

These reduplications, whatever their cause, are synchronous with definite phases of respiratory movement. I have elsewhere pointed out[14] that a splitting of the second sound at the base of the heart, beginning early in inspiration and increasing in interval to the end of the movement, is a perfectly normal phenomenon. Reduplication of the first sound of the heart at the apex, although somewhat less frequent, is still extremely common. Potain long ago showed that this doubling is usually confined to the end of the period of expiration. I have already discussed this subject at length, concluding that reduplications of the first sound of the heart are a sign of somewhat excessive, but still physiological, heart strain.

Clinical observation shows that the reduplication of the first sound at the apex is usually heard only in the erect position. When the subject lies supine the reduplication disappears and is commonly replaced by a soft systolic murmur that might easily escape detection. I have ventured the opinion that the reduplication itself is due usually to a late contraction of the papillary muscles of the left ventricle causing a corresponding and isolated sound of valve-tension, and that *the systolic murmur in the recumbent position is due to a real valvular insufficiency from imperfect papillary contraction.* Although the evidences for this conclusion are far from satisfactory, they seem to harmonize from different points of view. If crucial proof of the correctness of the suggestion can ever be furnished, a common "safety-valve" action of the mitral mechanism will have been established. The *a priori* probability that the right ventricle, at least, is provided with the capacity for a safety outflow within the range of physiological action is greatly strengthened by the clinical study of persons who manifestly suffer from functional insufficiency of the heart and in whom tracings of the jugular pulse furnish the most constant objective sign of the cardiac weakness.

---

[13] Foster's Physiology, 4th ed., 364.

[14] The Clinical Significance of Reduplication of the Heart Sounds. AMER. JOUR. MED. SCI., 1898, cxvi.

We turn now from a consideration of the problematical functional insufficiency of the auriculoventricular valves, the purpose of which in the economy must be to save the ventricles from overstrain, to a similar provision looking to the safety of the auricles. Admitting that the so-called "negative" venous pulse may at times have its origin in mere rhythmic intermission and acceleration of blood flow into the right auricle, there seems in many cases no doubt of the truth of Mackenzie's contention that even the "negative" or "auricular" form of venous pulse represents imperfect closure of the mouths of the great veins at the time of auricular systole, allowing regurgitation of blood from the contracting auricle. The important conclusion, deduced by Mackenzie from his clinical studies, that the existence of such a venous pulse always postulates a "limitation of the field of response" of the heart, almost necessarily carries with it the corrollary that even the lighter forms of venous pulse are manifestations of a *compensation* through which overstrain of the delicate auricle is prevented through backflow into the capacious "venous reservoir." A consideration of the conditions in compensated mitral disease leaves little doubt that the left auricle enjoys, at least, as efficient a protective outlet as the right.

In conclusion, while admitting the lack of the essential crucial evidence for the positions assumed in this paper, the physician in his practice must often act with less secure support. Strength has been added, I trust, to the conception that regurgitation from the auricles, thus preventing overstrain, is an occurrence so frequent that it cannot be regarded as a pathological event. Also, the indications are unmistakable that regurgitation from one or both ventricles, not caused by excessive dilatation of the auriculoventricular rings, but due to valvular insufficiency produced in another way, is an early and habitual sign of heart weakness. The therapeutic importance of the recognition of such signs of enfeeblement hardly needs an advocate.